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 FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:23:22 ON 03 JAN 2005
L1         94548 S WOLFFE?/AU OR COLLINGWOOD?/AU OR COX?/AU OR CASE?/AU OR JARVI
L2          5881 S CHROMATIN (P) REMODEL?
L3         86025 S FUSION (S) (PROTEIN OR CONSTRUCT OR NUCLEOTIDE OR PEPTIDE)
L4          1946 S "ZINC FINGER" (P) "BINDING DOMAIN"
L5         24358 S ("ZINC FINGER" OR DNA) (P) "BINDING DOMAIN"
L6         19860 S (CHROMATIN OR HISTONE) (P) (METHYLASE OR TRANSFERASE OR KINAS
L7           111 S L1 AND L2
L8            78 S L7 NOT PY>=2001
L9            34 DUP REM L8 (44 DUPLICATES REMOVED)
L10            0 S L9 AND L3
L11            1 S L9 AND L4
L12            2 S L9 AND L6
L13          111 S L2 AND L3
L14            2 S L13 AND L4
L15            2 S L13 AND L4
L16           17 S L13 AND L5
L17            7 DUP REM L16 (10 DUPLICATES REMOVED)
L18            1 S L17 NOT PY>=2001
L19            2 S L9 AND L5
L20            2 DUP REM L19 (0 DUPLICATES REMOVED)
L21           17 S L2 AND L3 AND L5
L22            3 S L21 NOT PY>=2001
L23            1 DUP REM L22 (2 DUPLICATES REMOVED)
L24          428 S L6 AND L3
L25           20 S L24 AND L5
L26            4 S L25 NOT PY>=2001
L27            2 DUP REM L26 (2 DUPLICATES REMOVED)
L28       307528 S ERYTHROPOIETIN OR ANDROGEN OR PPAR OR P16 OR P53 OR PRB OR DY
L29         2041 S L28 AND L5
L30          130 S L29 AND L3
L31           96 S L30 NOT PY>=2001
L32            0 S L31 AND CHROMATIN
L33            0 S L31 AND L1
L34            0 S L31 AND L6
L35           40 DUP REM L31 (56 DUPLICATES REMOVED)
L36            0 S L35 AND REMODEL?
L37            0 S L35 AND HISTONE
L38            0 S L35 AND "DNA PACKAGING"
L39          227 S L28 AND L2
L40           69 S L39 NOT PY>=2001
L41           27 DUP REM L40 (42 DUPLICATES REMOVED)
L42          194 S 41 AND L5
L43           11 S L42 AND L3
L44            7 DUP REM L43 (4 DUPLICATES REMOVED)
L45         1086 S 41 AND L3
L46            0 S L41 AND FUSION
L47           14 S L41 AND REGULATION
L48           14 DUP REM L47 (0 DUPLICATES REMOVED)

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ANSWER 2 OF 2 MEDLINE on STN
ACCESSION NUMBER: 2000179856 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10713069
TITLE: Two distinct nucleosome alterations characterize **chromatin remodeling** at the Saccharomyces cerevisiae ADH2 promoter.
AUTHOR: Di Mauro E; Kendrew S G; **Caserta M**
CORPORATE SOURCE: Centro di Studio per gli Acidi Nucleici, Consiglio Nazionale delle Ricerche, Universita "La Sapienza," P.le Aldo Moro 5, 00185 Rome, Italy.
SOURCE: Journal of biological chemistry, (2000 Mar 17) 275 (11) 7612-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000421
Last Updated on STN: 20030227
Entered Medline: 20000412

AB Glucose depletion derepresses the Saccharomyces cerevisiae ADH2 gene; this metabolic change is accompanied by **chromatin** structural modifications in the promoter region. We show that the ADR6/SWI1 gene is not necessary for derepression of the wild type chromosomal ADH2, whereas the transcription factor Adr1p, which regulates several S. cerevisiae functions, plays a major role in driving nucleosome reconfiguration and ADH2 expression. When we tested the effect of individual domains of the regulatory protein Adr1p on the **chromatin** structure of ADH2, a **remodeling** consisting of at least two steps was observed. Adr1p derivatives were analyzed in derepressing conditions, showing that the Adr1p **DNA binding domain** alone causes an alteration in **chromatin** organization in the absence of transcription. This alteration differs from the **remodeling** observed in the presence of the Adr1p activation domain when the promoter is transcriptionally active.

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ANSWER 1 OF 1 MEDLINE on STN
ACCESSION NUMBER: 1999110097 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9894806
TITLE: Recruitment of the RNA polymerase II holoenzyme and its implications in gene regulation.
AUTHOR: Barberis A; Gaudreau L
CORPORATE SOURCE: Institute of Molecular Biology, University of Zurich, Switzerland.
SOURCE: Biological chemistry, (1998 Dec) 379 (12) 1397-405. Ref: 82
Journal code: 9700112. ISSN: 1431-6730.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990317

AB In yeast cells, interaction between a **DNA**-bound protein and a single component of the RNA polymerase II (poIII) holoenzyme is sufficient to recruit the latter to a promoter and thereby activate gene transcription. Here we review results which have suggested such a simple mechanism for how genes can be turned on. The series of experiments which eventually led to this model was originally instigated by studying gene expression in a yeast strain which carries a point mutation in Gal11, a component of the poIII holoenzyme. In cells containing this mutant protein termed Gal11P, a derivative of the transcriptional activator Gal4 devoid of any classical activating region is turned into a strong activator. This activating function acquired by an otherwise silent **DNA**-binding protein is solely due to a novel and fortuitous interaction between Gal11P and a fragment of the Gal4 dimerization region generated by the P mutation. The simplest explanation for these results is that tethering Gal11 to **DNA** recruits the poIII holoenzyme and, consequently, activates gene transcription. Transcription factors that are believed not to be integral part of the poIII holoenzyme but are nevertheless required for this instance of gene activation, e.g. the TATA-binding TFIID complex, may bind **DNA** cooperatively with the holoenzyme when recruited to a promoter, thus forming a complete poIII preinitiation complex. One prediction of this model is that recruitment of the entire poIII transcription complex and consequent gene activation can be achieved by tethering different components to **DNA**. Indeed, **fusion** of a **DNA-binding domain** to a variety of poIII holoenzyme components and TFIID subunits leads to activation of genes bearing the recognition site for the **DNA**-binding **protein**. These results imply that accessory factors, which are required to remove or modify nucleosomes do not need to be directly contacted by activators, but can rather be engaged in the activation process when the poIII complex is recruited to **DNA**. In fact, recruitment of the poIII holoenzyme suffices to **remodel** nucleosomes at the PHO5 promoter and presumably at many other promoters. Other events in the process of gene expression following recruitment of the transcription complex, e.g. initiation, promoter clearance, elongation and termination, could unravel as a consequence of the recruitment step and the formation of an active preinitiation complex on **DNA**. This view does not exclude the possibility that classical activators also act directly on **chromatin remodeling** and post-recruitment steps to regulate gene expression.

L11 ANSWER 1 OF 1 MEDLINE on STN
ACCESSION NUMBER: 2001038274 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10913152
TITLE: Synthetic zinc finger transcription factor action at an endogenous chromosomal site. Activation of the human erythropoietin gene.
AUTHOR: Zhang L; Spratt S K; Liu Q; Johnstone B; Qi H; Raschke E E; Jamieson A C; Rebar E J; **Wolffe A P; Case C C**
CORPORATE SOURCE: Sangamo BioSciences Inc., Point Richmond Tech Center, Richmond, California 94804, USA.
SOURCE: Journal of biological chemistry, (2000 Oct 27) 275 (43) 33850-60.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001124

AB We have targeted the activation of an endogenous chromosomal locus including the human erythropoietin gene using synthetic transcription factors. These transcription factors are targeted to particular DNA sequences in the 5'-flanking region of the erythropoietin gene through engineering of a **zinc finger** DNA **binding domain**. The DNA **binding domain** is linked to a VP16 transcriptional activation domain. We find that these synthetic transcription factors invariably activate transiently transfected templates in which sequences within the 5' flank of the erythropoietin gene are fused to a luciferase reporter. The efficiency of activation under these circumstances at a defined site is dependent on DNA binding affinity. In contrast, only a subset of these same **zinc finger** proteins is able to activate the endogenous chromosomal locus. The activity of these proteins is influenced by their capacity to gain access to their recognition elements within the **chromatin** infrastructure. **Zinc finger** transcription factors will provide a powerful tool to probe the determinants of **chromatin** accessibility and **remodeling** within endogenous chromosomal loci.

ANSWER 1 OF 2 MEDLINE on STN
ACCESSION NUMBER: 97076150 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8918467
TITLE: **Remodeling** somatic nuclei in Xenopus laevis egg extracts: molecular mechanisms for the selective release of histones H1 and H1(0) from **chromatin** and the acquisition of transcriptional competence.
AUTHOR: Dimitrov S; **Wolffe A P**
CORPORATE SOURCE: Laboratoire d'Etudes de la Differentiation et de l'Adherence cellulaire, UMR 5538, Centre National de la Recherche Scientifique, Institut Albert Bonniot, La Tronche, France.
SOURCE: EMBO journal, (1996 Nov 1) 15 (21) 5897-906.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19970108

AB The molecular mechanisms responsible for the **remodeling** of entire somatic erythrocyte nuclei in Xenopus laevis egg cytoplasm have been examined. These transitions in chromosomal composition are associated with the capacity to activate new patterns of gene expression and the re-acquisition of replication competence. Somatic linker **histone** variants H1 and H1 (0) are released from **chromatin** in egg cytoplasm, whereas the oocyte-specific linker **histone** B4 and HMG1 are efficiently incorporated into **remodeled chromatin**. **Histone** H1 (0) is released from **chromatin** preferentially in comparison with **histone** H1. Core histones H2A and H4 in the somatic nucleus are phosphorylated during this **remodeling** process. These transitions recapitulate the chromosomal environment found within the nuclei of the early Xenopus embryo. **Phosphorylation** of somatic linker **histone** variants is demonstrated not to direct their release from **chromatin**, nor does direct competition with cytoplasmic stores of linker **histone** B4 determine their release. However, the molecular chaperone nucleoplasmin does have an important role in the selective removal of linker histones from somatic nuclei. For Xenopus erythrocyte nuclei, this disruption of **chromatin** structure leads to activation of the 5S rRNA genes. These results provide a molecular explanation for the **remodeling** of **chromatin** in Xenopus egg cytoplasm and indicate the capacity of molecular chaperones to disrupt a natural chromosomal environment, thereby facilitating transcription.

L12 ANSWER 2 OF 2 MEDLINE on STN
ACCESSION NUMBER: 94321419 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8045925
TITLE: **Remodeling** sperm **chromatin** in Xenopus laevis egg extracts: the role of core **histone phosphorylation** and linker **histone** B4 in **chromatin** assembly.
AUTHOR: Dimitrov S; Dasso M C; **Wolffe A P**
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland 20892.
SOURCE: Journal of cell biology, (1994 Aug) 126 (3) 591-601.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940909
Last Updated on STN: 19940909
Entered Medline: 19940826

AB We find that the remodeling of the condensed Xenopus laevis sperm nucleus into the paternal pronucleus in egg extracts is associated with **phosphorylation** of the core histones H2A, H2A.X and H4, and uptake of a linker **histone** B4 and a HMG 2 protein. **Histone** B4 is required for the assembly of chromatosome structures in the pronucleus. However neither B4 nor core **histone** **phosphorylation** are required for the assembly of spaced nucleosomal arrays. We suggest that the spacing of nucleosomal arrays is determined by interaction between adjacent **histone** octamers under physiological assembly conditions.

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ANSWER 4 OF 40 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2000214797 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10750018
TITLE: Association of the Ku autoantigen/**DNA**-dependent protein kinase holoenzyme and poly(ADP-ribose) polymerase with the **DNA binding domain** of progesterone receptors.
AUTHOR: Sartorius C A; Takimoto G S; Richer J K; Tung L; Horwitz K B
CORPORATE SOURCE: Department of Medicine, University of Colorado Health Sciences Center, Denver, Colorado 80262, USA.. Carol.Sartorius@uchsc.edu
CONTRACT NUMBER: CA26869 (NCI)
DK48238 (NIDDK)
SOURCE: Journal of molecular endocrinology, (2000 Apr) 24 (2) 165-82.
Journal code: 8902617. ISSN: 0952-5041.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000518
Last Updated on STN: 20030218
Entered Medline: 20000508

AB Ligand-activated progesterone receptors (PR) bind to **DNA** at specific progesterone response elements by means of a **DNA binding domain** (DBD(PR)) containing two highly conserved zinc fingers. **DNA**-bound PRs regulate transcription via interaction with other nuclear proteins and transcription factors. We have now identified four HeLa cell nuclear proteins that copurify with a glutathionine-S-transferase-human DBD(PR)**fusion protein** . Microsequence and immunoblot analyses identified one of these proteins as the 113 kDa poly(ADP-ribose) polymerase. The three other proteins were identified as subunits of the **DNA**-dependent protein kinase (**DNA**-PK) holoenzyme: its **DNA** binding regulatory heterodimers consisting of Ku70 and Ku86, and the 460 kDa catalytic subunit, **DNA**-PK(CS). **DNA**-PK that was 'pulled-down' by DBD(PR) on the affinity resin was able to (1) autophosphorylate Ku70, Ku86, and **DNA**-PK(CS), (2) transphosphorylate DBD(PR), and (3) phosphorylate a **DNA**-PK-specific **p53** peptide substrate. **DNA**-PK was also able to associate with the DBD of the yeast activator GAL4. However, neither a PR DBD mutant lacking a structured first **zinc finger** (DBD(CYS)) nor the core DBD of the estrogen receptor (DBD(ER)) copurified **DNA**-PK, suggesting the interaction is not non-specific for DBDs. Lastly, we found that **DNA**-PK copurified with full-length human PR transiently expressed in HeLa cells, suggesting that the human PR/**DNA**-PK complex can assemble in vivo. These data show that **DNA**-PK and DBD(PR) interact, that DBD(PR) is a phosphorylation substrate of **DNA**-PK, and suggest a potential role for **DNA**-PK in PR-mediated transcription.

L35 ANSWER 5 OF 40 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2000092325 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10628744
TITLE: Protein inhibitor of activated STAT-1 (signal transducer and activator of transcription-1) is a nuclear receptor coregulator expressed in human testis.
AUTHOR: Tan J; Hall S H; Hamil K G; Grossman G; Petrusz P; Liao J; Shuai K; French F S
CORPORATE SOURCE: Department of Pediatrics, University of North Carolina

School of Medicine, Chapel Hill 27599-7500, USA.
CONTRACT NUMBER: AI 43438 (NIAID)
R37 HD-04466 (NICHD)
T32 HD-07315 (NICHD)
+
SOURCE: Molecular endocrinology (Baltimore, Md.), (2000 Jan) 14 (1) 14-26.
Journal code: 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF167160
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000204
Last Updated on STN: 20000204

NSWER 13 OF 14 MEDLINE on STN
ACCESSION NUMBER: 97246560 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9092798
TITLE: Two distinct mechanisms elicit **androgen**-dependent expression of the mouse sex-limited protein gene.
AUTHOR: Nelson S A; Robins D M
CORPORATE SOURCE: Department of Human Genetics, University of Michigan Medical School, Ann Arbor 48109-0618, USA.
CONTRACT NUMBER: GM-31546 (NIGMS)
P30-HD-18258 (NICHD)
SOURCE: Molecular endocrinology (Baltimore, Md.), (1997 Apr) 11 (4) 460-9.
Journal code: 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970908
Last Updated on STN: 19970908
Entered Medline: 19970826

AB The mouse sex-limited protein (Slp) gene is expressed in liver and kidney of adult males and is testosterone-inducible in females, indicative of **androgen** dependence. Analysis of mRNA levels and **chromatin** configuration reveals that this **androgen regulation** is achieved by distinct means in the two tissues. In the liver, Slp expression requires pituitary function, and specifically, as shown by others, a pulsatile pattern of GH secretion that is itself determined by **androgen**. After hypophysectomy, Slp synthesis cannot be reestablished in liver by testosterone, although mRNA decline can be slowed. In contrast, in the kidney Slp mRNA is directly induced by **androgen** in hypophysectomized mice. In vivo footprinting was used to examine the role of the Slp enhancer, which directs **androgen** -specific transcription in transfection and contains a factor-binding site, FPIV, whose protection in vivo has been correlated with Slp expression. In kidney, FPIV was protected in intact males and

ANSWER 20 OF 40	MEDLINE on STN DUPLICATE 15
ACCESSION NUMBER:	96189091 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 8628276
TITLE:	Adenovirus E1A proteins inhibit activation of transcription by **p53**.
AUTHOR:	Steegenga W T; van Laar T; Riteco N; Mandarino A; Shvarts A; van der Eb A J; Jochemsen A G
CORPORATE SOURCE:	Laboratory of Molecular Carcinogenesis, Sylvius Laboratories, Leiden University, The Netherlands.
SOURCE:	Molecular and cellular biology, (1996 May) 16 (5) 2101-9. Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199606
ENTRY DATE:	Entered STN: 19960708 Last Updated on STN: 19970203 Entered Medline: 19960621

L35 ANSWER 10 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1998028738 EMBASE

TITLE: Intermolecular NH2-/carboxyl-terminal interactions in **androgen** receptor dimerization revealed by mutations that cause **androgen** insensitivity.

AUTHOR: Langley E.; Kemppainen J.A.; Wilson E.M.

CORPORATE SOURCE: E.M. Wilson, Lab. for Reproductive Biology, Medical Scineces Research Bldg., University of North Carolina, Chapel Hill, NC 27599, United States. emw@med.unc.edu

SOURCE: Journal of Biological Chemistry, (1998) 273/1 (92-101). Refs: 47
ISSN: 0021-9258 CODEN: JBCHA3

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 029 Clinical Biochemistry

L35 ANSWER 37 OF 40 MEDLINE on STN DUPLICATE 26

ACCESSION NUMBER: 92347601 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1639214
TITLE: Expression and characterization of full-length and partial human **androgen** receptor fusion proteins. Implications for the production and applications of soluble steroid receptors in Escherichia coli.
AUTHOR: Roehrborn C G; Zoppi S; Gruber J A; Wilson C M; McPhaul M J
CORPORATE SOURCE: Department of Internal Medicine, The University of Texas Southwestern Medical Center, Dallas 75235-8857.
CONTRACT NUMBER: DK03892 (NIDDK)
SOURCE: Molecular and cellular endocrinology, (1992 Mar) 84 (1-2) 1-14.
Journal code: 7500844. ISSN: 0303-7207.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920911
Last Updated on STN: 19980206

	U	Document ID	Title
1	X	US 20040204345 A1	Modulation of endogenous gene expression in cells
2	X	US 20030134318 A1	Methods of using randomized libraries of zinc finger proteins for the identification of gene function
3	X	US 20030021776 A1	Regulation of angiogenesis with zinc finger proteins
4	X	US 6610489 B2	Pharmacogenomics and identification of drug targets by reconstruction of signal transduction pathways based on sequences of accessible regions
5	X	US 6511808 B2	Methods for designing exogenous regulatory molecules
6	X	US 6503717 B2	Methods of using randomized libraries of zinc finger proteins for the identification of gene function
7	X	WO 200244386 A	Regulating expression of gene by contacting cell with regulatory molecule comprising DNA-binding domain targeted to sequence within accessible region of cellular chromatin associated with a gene, and functional domain
8	X	WO 200244376 A	Modulating expression of gene, comprises contacting region of DNA in cellular chromatin with fusion molecule containing DNA binding domain and insulator domain, that binds to binding site in cellular chromatin

	U	Document ID	Title
9	X	WO 200226960 A	Compartmentalizing a region of interest in cellular chromatin which facilitates the modulation of the expression of a gene comprises contacting the gene with a composition comprising a localization domain and a DNA binding domain
10	X	US 20020094968 A	Gene regulation and nuclear programming for cloning and modulation of cellular differentiation and dedifferentiation, comprises contacting a somatic nucleus with a composition containing ISWI, which remodels the somatic nucleus
11	X	WO 200183819 A	Designing exogenous regulatory molecules for regulating a gene of interest comprises preparation based on identified regulatory sequence elements from accessible regions of chromatin
12	X	US 20020115215 A	Modification of chromatin structure for facilitating transcription, replication and repair, comprises contacting chromatin with fusion molecule comprising DNA binding domain and component of a chromatin remodeling complex

	U	Document ID	Title
13	X	US 20020064802 A	Binding an exogenous molecule (EM) to a binding site located within a region of interest in chromatin, useful for modulating gene expression, by identifying an EM target site within an accessible region and introducing the EM into the cell
14	X	US 20020127559 A	Identification of drugs for the treatment of cancer, cardiovascular disease or osteoporosis comprises targeting gene regions of interest of the transduction pathway
15	X	US 20020081603 A	Isolating a collection of polynucleotides corresponding to accessible regions of cellular chromatin, for generating a polynucleotide library and database, comprises using a probe or enzyme

	Document ID	Title
1	US 20040204345 A1	Modulation of endogenous gene expression in cells
2	US 20030232781 A1	Modulation of gene expression using insulator binding proteins
3	US 20030190664 A1	Pharmacogenomics and idenitfication of drug targets by reconstruction of signal transduction pathways based on sequences of accessible regions
4	US 20030129603 A1	Databases of regulatory sequences; methods of making and using same
5	US 20030082552 A1	Modulation of gene expression using localization domains
6	US 20030049649 A1	Targeted modification of chromatin structure
7	US 20030044404 A1	Regulation of angiogenesis with zinc finger proteins
8	US 20030021776 A1	Regulation of angiogenesis with zinc finger proteins
9	US 20020160940 A1	Modulation of endogenous gene expression in cells
10	US 20020127559 A1	Pharmacogenomics and identification of drug targets by reconstruction of signal transduction pathways based on sequences of accessible regions
11	US 20020115215 A1	Targeted modification of chromatin structure
12	US 20020094968 A1	Nuclear reprogramming using IWSI and related chromatin remodeling ATPases
13	US 20020081603 A1	Databases of regulatory sequences; methods of making and using same
14	US 20020076711 A1	Methods for designing exogenous regulatory molecules

	Document ID	Title
15	US 20020064802 A1	Methods for binding an exogenous molecule to cellular chromatin
16	US 6610489 B2	Pharmacogenomics and identification of drug targets by reconstruction of signal transduction pathways based on sequences of accessible regions
17	US 6511808 B2	Methods for designing exogenous regulatory molecules
18	US 20020160940 A	Modulating the expression of an endogenous gene in a cell, e.g. for treating genetic diseases and developing plants with altered phenotypes, comprises contacting a target site in the gene with a zinc finger protein
19	WO 200244376 A	Modulating expression of gene, comprises contacting region of DNA in cellular chromatin with fusion molecule containing DNA binding domain and insulator domain, that binds to binding site in cellular chromatin
20	US 20020115215 A	Modification of chromatin structure for facilitating transcription, replication and repair, comprises contacting chromatin with fusion molecule comprising DNA binding domain and component of a chromatin remodeling complex